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#24	Search hama and muc-1	15:57:25	4
#23	Search hama and ca 125	15:57:11	16
#22	Search xenotypic antibody and T cytotoxic Limits: Publication Date to 1998/3/20	14:16:11	0
#21	Search xenotypic antibody and T helper Limits: Publication Date to 1998/3/20	14:15:45	1
#20	Search xenotypic antibody and T lymphocyte Limits: Publication Date to 1998/3/20	14:15:12	0
#19	Search xenotypic antibody and T cell Limits: Publication Date to 1998/3/20	14:14:47	1
#18	Search prostate specific antigen and idiotype Limits: Publication Date to 1998/3/20	14:12:03	0
#17	Search prostate specific antigen and anti-idiotype Limits: Publication Date to 1998/3/20	14:11:56	0
#14	Search anti-idiotypic antibody and prostate antigen Limits: Publication Date to 1998/3/20	14:09:51	4
#13	Search anti-idiotypic antibody and prostate Limits: Publication Date to 1998/3/20	14:09:15	6
#12	Search anti-idiotypic antibody and psa Limits: Publication Date to 1998/3/20	14:08:50	1
#10	Search anti-idiotypic antibody and MUC-1 Limits: Publication Date to 1998/3/20	14:07:05	7
#8	Search anti-idiotypic antibody and CA 125 Limits: Publication Date to 1998/3/20	14:04:30	25
#6	Search xenotypic antibody Limits: Publication Date to 1998/3/20	13:59:59	6
#5	Search xenotypic antibody and anti-idiotype Limits: Publication Date to 1998/3/20	13:59:43	0
#4	Search xenotypic antibody and psa Limits: Publication Date to 1998/3/20	13:58:46	0
#3	Search xenotypic antibody and MUC-1 Limits: Publication Date to 1998/3/20	13:58:37	0
#2	Search xenotypic antibody and CA 125 Limits: Publication Date to 1998/3/20	13:58:28	0
#1	Search xenotypic antibody and CA125 Limits: Publication Date to 1998/3/20	13:58:18	0

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AN/altarex: 5 patents.

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AN/altarex

PAT. NO. Title

- 1 [6,881,405](#)  [Reagents and methods for inducing an immune response to prostate specific antigen](#)
  - 2 [6,716,966](#)  [Therapeutic binding agents against MUC-1 antigen and methods for their use](#)
  - 3 [6,689,355](#)  [Therapeutic method and composition utilizing antigen-antibody complexation and presentation by dendritic cells](#)
  - 4 [6,241,985](#)  [Method and composition for reconfirming multi-epitopic antigens to initiate an immune response](#)
  - 5 [6,086,873](#)  [Therapeutic composition and method of treatment](#)
- 

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## US PATENT & TRADEMARK OFFICE

### PATENT APPLICATION FULL TEXT AND IMAGE DATABASE



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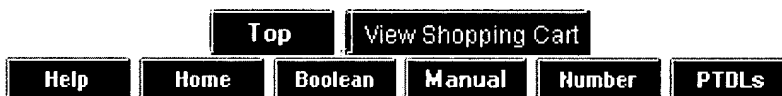
AN/altarex: 3 applications.

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PUB. APP. NO. Title

- |   |                             |  |
|---|-----------------------------|--|
| 1 | <a href="#">20050260208</a> | <a href="#">Binding agents and their use in targeting tumor cells</a>                      |
| 2 | <a href="#">20050063976</a> | <a href="#">Combination therapy for treating disease</a>                                   |
| 3 | <a href="#">20050048059</a> | <a href="#">Therapeutic binding agents against MUC-1 antigen and methods for their use</a> |



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## Refine Search

hama AND ca125 and "idiotypic antibody"

PAT. NO.	Title
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1 6,241,985  Method and composition for reconforming multi-epitopic antigens to initiate an immune response

**2 6,086,873** **¶ Therapeutic composition and method of treatment**

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**((hama AND "prostate specific antigen") AND antibody) AND anti-idiotypic): 4 patents.**

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## Refine Search

# hama AND "prostate specific antigen" and antibody ai

PAT. NO.	Title
----------	-------

## 1 6,689,744 Notch receptor agonists and uses

**2 6,331,402**  **Reduction of interference of immunoassays by substances derived from the framework regions of antibodies**

3 6,274,118 Localization and therapy of non-prostatic endocrine cancer with agents directed against prostate specific antigen

4 6,068,830 Localization and therapy of non-prostatic endocrine cancer with agents directed against prostate specific antigen

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PUB. APP. NO. Title

- |   |                             |  |
|---|-----------------------------|--|
| 1 | <a href="#">20060159688</a> | <a href="#">Method for diagnosing efficacy of xenotypic antibody therapy</a>                                   |
| 2 | <a href="#">20050169932</a> | <a href="#">Uses of monoclonal antibody 8h9</a>  |
| 3 | <a href="#">20050031619</a> | <a href="#">Therapeutic compositions that alter the immune response</a>  |
| 4 | <a href="#">20030103963</a> | <a href="#">Uses of monoclonal antibody 8H9</a>  |
| 5 | <a href="#">20020102264</a> | <a href="#">Uses of monoclonal antibody 8H9</a>  |
| 6 | <a href="#">20020048586</a> | <a href="#">THERAPEUTIC COMPOSITIONS THAT ALTER THE IMMUNE RESPONSE</a>  |
| 7 | <a href="#">20020022235</a> | <a href="#">Method for diagnosing efficacy of xenotypic antibody therapy</a>                                   |
| 8 | <a href="#">20010036457</a> | <a href="#">Method and composition for reconfirming multi-epitopic antigens to initiate an immune response</a> |



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PUB. APP. NO. Title

51	<a href="#">20020022235</a>	<a href="#">Method for diagnosing efficacy of xenotypic antibody therapy</a>
52	<a href="#">20010036457</a>	<a href="#">Method and composition for reconfirming multi-epitopic antigens to initiate an immune response</a>
53	<a href="#">20010021381</a>	<a href="#">Anti-TNFalpha antibodies in therapy of asthma</a>





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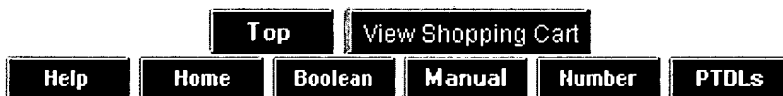
Hits 1 through 10 out of 10

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Refine Search hama AND "idiotypic antibody" and "prostate specific

PUB. APP. NO. Title

- 1 [20060159688](#) [Method for diagnosing efficacy of xenotypic antibody therapy](#)
- 2 [20060147375](#) [Antibodies and related molecules that bind to PSCA proteins](#)
- 3 [20050221400](#) [Antibodies and related molecules that bind to PSCA proteins](#)
- 4 [20050142539](#) [Targeted ligands](#)
- 5 [20050118164](#) [Targeted ligands](#)
- 6 [20050069549](#) [Targeted ligands](#)
- 7 [20050031619](#) [Therapeutic compositions that alter the immune response](#)
- 8 [20040019915](#) [Nucleic acid and corresponding protein entitled 213P1F11 useful in treatment and detection of cancer](#)
- 9 [20020048586](#) [THERAPEUTIC COMPOSITIONS THAT ALTER THE IMMUNE RESPONSE](#)
- 10 [20020022235](#) [Method for diagnosing efficacy of xenotypic antibody therapy](#)



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L1 12 XENOTYPIC ANTIBODY

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L2 12621 IDIOTYPE OR IDIOTYPIC

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=> d l4 bib abs 1-2

L4 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2006 ACS on STN

Full Text	Citing References
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AN 2006:708190 CAPLUS

DN 145:123046

TI Method for diagnosing efficacy of **xenotypic antibody** therapy

IN Madiyalakan, Ragupathy; Noujaim, Antoine A.; Baum, Richard P.

PA Can.

SO U.S. Pat. Appl. Publ., 15 pp., Cont.-in-part of U.S. Ser. No. 871,339.

CODEN: USXXCO

DT Patent

LA English

FAN.CNT 9

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	<u>US 2006159688</u>	A1	20060720	<u>US 2004-824554</u>	20040414
	<u>WO 9742973</u>	A1	19971120	<u>WO 1996-IB461</u>	19960515
	W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN				
	RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
	<u>JP 2001055341</u>	A2	20010227	<u>JP 2000-200702</u>	19960515
	<u>NZ 503032</u>	A	20011130	<u>NZ 1996-503032</u>	19960515
	<u>EP 1297846</u>	A1	20030402	<u>EP 2002-18963</u>	19960515
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, AL				
	<u>PT 910407</u>	T	20030731	<u>PT 1996-913660</u>	19960515
	<u>ES 2193240</u>	T3	20031101	<u>ES 1996-913660</u>	19960515
	<u>US 6241985</u>	B1	20010605	<u>US 1998-913290</u>	19980320
	<u>US 2002022235</u>	A1	20020221	<u>US 2001-779439</u>	20010208
	<u>US 2001036457</u>	A1	20011101	<u>US 2001-871339</u>	20010531
	<u>JP 2004002481</u>	A2	20040108	<u>JP 2003-315495</u>	20030908
	<u>AU 2006203074</u>	A1	20060810	<u>AU 2006-203074</u>	20060718
PRAI	<u>WO 1996-IB461</u>	A	19960515		

<u>US 1998-913290</u>	A1	19980320
<u>US 2000-181008P</u>	P	20000208
<u>US 2001-779439</u>	B1	20010208
<u>US 2001-871339</u>	A2	20010531
<u>CA 1996-2253602</u>	A	19960515
<u>EP 1996-913660</u>	A3	19960515
<u>JP 1997-540681</u>	A3	19960515
<u>JP 2000-200702</u>	A3	19960515
<u>NZ 1996-332588</u>	A1	19960515
<u>US 2000-201868P</u>	P	20000504
<u>AU 2001-40982</u>	A3	20010208

AB The authors disclose methods and compns. for intiating and/or enhancing an immune response by contacting an antibody with a sol. antigen. The resulting immune complex generates an enhanced immune response to the antigen. In one example, the authors demonstrate an enhanced T-cell response to an immune complex of sol. CA125 and the B43.13 monoclonal antibody.

L4 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2006 ACS on STN

Full Text	Citing References
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AN 2001:598293 CAPLUS

DN 135:179715

TI Method for diagnosing efficacy of **xenotypic antibody** therapy

IN Noujaim, Antoine

PA Altarex Corp., Can.

SO PCT Int. Appl., 28 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 9

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
<u>PI</u>	<u>WO 2001059452</u>	A2	20010816	<u>WO 2001-IB423</u>	20010208
	<u>WO 2001059452</u>	A3	20020718		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	<u>JP 2001055341</u>	A2	20010227	<u>JP 2000-200702</u>	19960515
	<u>NZ 503032</u>	A	20011130	<u>NZ 1996-503032</u>	19960515
	<u>EP 1297846</u>	A1	20030402	<u>EP 2002-18963</u>	19960515
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, AL				
	<u>PT 910407</u>	T	20030731	<u>PT 1996-913660</u>	19960515
	<u>ES 2193240</u>	T3	20031101	<u>ES 1996-913660</u>	19960515
	<u>CA 2399067</u>	AA	20010816	<u>CA 2001-2399067</u>	20010208
	<u>AU 2001040982</u>	A5	20010820	<u>AU 2001-40982</u>	20010208
	<u>EP 1254374</u>	A2	20021106	<u>EP 2001-912066</u>	20010208
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
	<u>JP 2003522774</u>	T2	20030729	<u>JP 2001-558731</u>	20010208
	<u>JP 2004002481</u>	A2	20040108	<u>JP 2003-315495</u>	20030908
	<u>AU 2006203074</u>	A1	20060810	<u>AU 2006-203074</u>	20060718
<u>PRAI</u>	<u>US 2000-181008P</u>	P	20000208		
	<u>US 2000-201868P</u>	P	20000504		
	<u>EP 1996-913660</u>	A3	19960515		
	<u>JP 1997-540681</u>	A3	19960515		
	<u>JP 2000-200702</u>	A3	19960515		
	<u>NZ 1996-332588</u>	A1	19960515		
	<u>AU 2001-40982</u>	A3	20010208		

WO 2001-IB423 W 20010208

AB The invention provides methods for diagnosing the efficacy of a patient to **xenotypic antibody** therapy which include (1) measuring the level of an antibody produced by a patient that specifically binds to a **xenotypic antibody** after administration of the **xenotypic antibody** to the patient; (2) measuring the level of an anti-idiotypic antibody produced by a patient that specifically binds to a **xenotypic antibody** after administration of the **xenotypic antibody** to the patient; (3) measuring the level of an antibody produced by a patient that specifically binds to a target antigen of a **xenotypic antibody** after administration of a **xenotypic antibody** to the patient; and (4) measuring the level of a T cell response produced by a patient to a target antigen of the **xenotypic antibody** after administration of a **xenotypic antibody** to the patient. In the methods of the invention, an increase in the level of antibody or T cell response produced by the patient after the administration of the **xenotypic antibody** relative to the level antibody or T cell response produced by the patient prior to the administration of the **xenotypic antibody** is indicative of a favorable diagnosis of efficacy.

=> s xenotype and antibody

L5 3 XENOTYPE AND ANTIBODY

=> duplicate remove l5

DUPLICATE PREFERENCE IS 'CAPLUS, BIOTECHDS, ESBIODASE'

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L6 1 DUPLICATE REMOVE L5 (2 DUPLICATES REMOVED)

=> d l6 bib abs 1

L6 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 1

	Full Text	Citing References
AN	2005:1294599	CAPLUS
DN	144:218935	
TI	Fluorescently tagged canine adenovirus via modification with protein IX-enhanced green fluorescent protein	
AU	Le, Long P.; Li, Jing; Ternovoi, Vladimir V.; Siegal, Gene P.; Curiel, David T.	
CS	Division of Human Gene Therapy, Departments of Medicine, Pathology and Surgery, University of Alabama at Birmingham, Birmingham, AL, 35294, USA	
SO	Journal of General Virology (2005), 86(12), 3201-3208 CODEN: JGVIAI; ISSN: 0022-1317	
PB	Society for General Microbiology	
DT	Journal	
LA	English	
AB	Canine adenovirus type 2 (CAV2) has become an attractive vector for gene therapy because of its non-pathogenicity and the lack of pre-existing neutralizing antibodies against this virus in the human population. Addnl., this vector has been proposed as a conditionally replicative adenovirus agent under the control of an osteocalcin promoter for evaluation in a syngeneic, immunocompetent canine model with spontaneous osteosarcoma. In this study, a CAV2 vector labeled with the fluorescent capsid fusion protein IX-enhanced green fluorescent protein (pIX-EGFP) was developed. Expression of the fluorescent fusion-protein label in infected cells with proper nuclear localization, and incorporation into virions, could be detected. The labeled virions could be visualized by fluorescence microscopy; this was applicable to the tracking of CAV2 infection, as well as localizing the distribution of the vector in tissues. Expression of pIX-EGFP could be exploited to detect the replication and spread of CAV2. These results indicate that pIX can serve as a platform for incorporation of heterologous proteins in the context of a canine adenovirus <b>xenotype</b> . It is believed that capsid-labeled CAV2 has utility for vector-development studies and for monitoring CAV2-based	

oncolytic adenovirus replication.

RE.CNT 45 THERE ARE 45 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> s xenogeneic antibody and  
MISSING TERM AFTER ANTIBODY AND  
Operators must be followed by a search term, L-number, or query name.

=> s xenogeneic antibody  
L7 79 XENOGENEIC ANTIBODY

=> s l7 and idiotype or anti-idiotypic  
L8 5894 L7 AND IDIOTYPE OR ANTI-IDIOTYPIC

=> s l7 and anti-idiotypic  
L9 8 L7 AND ANTI-IDIOTYPIC

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=> d l10 bib abs 1-8

L10 ANSWER 1 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN

	Full Text	Citing References
AN	2005:343313	CAPLUS
DN	142:461722	
TI	SDR grafting-a new approach to antibody humanization	
AU	Kashmiri, Syed V. S.; De Pascalis, Roberto; Gonzales, Noreen R.; Schlom, Jeffrey	
CS	Laboratory of Tumor Immunology and Biology, Center for Cancer Research, National Cancer Institute, National Institutes of Health, Bethesda, MD, 20892, USA	
SO	Methods (San Diego, CA, United States) (2005), 36(1), 25-34 CODEN: MTHDE9; ISSN: 1046-2023	
PB	Elsevier	
DT	Journal; General Review	
LA	English	
AB	<p>A review. A major impediment to the clin. utility of the murine monoclonal antibodies is their potential to elicit human anti-murine antibody (HAMA) response in patients. To circumvent this problem, murine antibodies have been genetically manipulated to progressively replace their murine content with the amino acid residues present in their human counterparts. To that end, murine antibodies have been humanized by grafting their complementarity detg. regions (CDRs) onto the variable light (VL) and variable heavy (VH) frameworks of human Ig mols., while retaining those murine framework residues deemed essential for the integrity of the antigen-combining site. However, the xenogeneic CDRs of the humanized antibodies may evoke <b>anti-idiotypic</b> (anti-Id) response in patients. To minimize the anti-Id response, a procedure to humanize <b>xenogeneic antibodies</b> has been described that is based on grafting, onto the human frameworks, only the specificity detg. residues (SDRs), the CDR residues that are most crucial in the antibody-ligand interaction. The SDRs are identified through the help of the database of the three-dimensional structures of the antigen-antibody complexes of known structures or by mutational anal. of the antibody-combining site. An alternative approach to humanization, which involves retention of more CDR residues, is based on grafting of the 'abbreviated' CDRs, the stretches of CDR residues that include all the SDRs. A procedure to assess the reactivity of the humanized antibody to sera from patients who had been administered the murine antibody has also been described.</p>	

RE.CNT 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD

## ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 2 OF 8 BIOTECHDS COPYRIGHT 2006 THE THOMSON CORP. on STN

Full  
Text

AN 2004-12683 BIOTECHDS  
 TI New antibodies 34B1, 26H7, and 5F4 that modulate T cell activity, useful for modulating an immune response, for treating graft versus host disease, sepsis or tumors, and for improving wound healing; monoclonal antibody preparation by hybridoma cell culture for disease therapy  
 AU BLUMBERG R S; BHAN A  
 PA BLUMBERG R S; BHAN A  
 PI US 2004047858 11 Mar 2004  
 AI US 2002-241369 11 Sep 2002  
 PRAI US 2002-241369 11 Sep 2002; US 2002-241369 11 Sep 2002  
 DT Patent  
 LA English  
 OS WPI: 2004-226167 [21]  
 AN 2004-12683 BIOTECHDS  
 AB DERWENT ABSTRACT:

NOVELTY - An antibody that modulates T cell activity selected from monoclonal antibody 34B1, 26H7, and 5F4 as produced by hybridoma 34B1, 26H7 and 5F4, respectively, deposited with ATCC (accession numbers not given), on September 4, 2002, or their antigen-binding fragment.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following: (1) a monoclonal antibody or its antigen-binding fragment that modulates T cell activity, and competes with the antibody defined above for binding to the N-domain of BGP (C-CAM1); (2) a hybridoma that produces a monoclonal antibody above; (3) a polynucleotide encoding at least a variable region of an immunoglobulin chain of any of the antibodies above; (4) a vector comprising the polynucleotide, optionally in combination with a polynucleotide that encodes the variable region of the other immunoglobulin chain of the antibody; (5) a host cell comprising a polynucleotide or vector; (6) a method for preparing an antibody or its functional fragment or immunoglobulin chain(s); (7) an antibody, its immunoglobulin chain or binding fragment encoded by a polynucleotide above, or obtainable by the method of (6); (8) a composition comprising any of the above antibody or antigen-binding fragment, polynucleotide, vector or cell; (9) a diagnostic composition comprising any of the above defined antibody, polynucleotide, vector or cell, and optionally reagents conventionally used in immuno- or nucleic acid based diagnostic methods; (10) a method of modulating the immune response in a subject by administering an antibody defined above; (11) a method of diagnosing and/or treatment of a disorder related to the neo-expression or malfunction of BGP(C-CAM1) by administering a ligand binding molecule comprising at least one CDR of an antibody above, or a corresponding **anti-idiotypic** anti-body; and (12) a method of targeting a therapeutic and/or diagnostic agent to a cell which expresses BGP(C-CAM1) or its fragment, by administering a ligand binding molecule comprising at least one CDR of an antibody above.

BIOTECHNOLOGY - Preferred Antibody: The monoclonal antibody or antigen-binding fragment is capable of inhibiting T cell proliferation in an allogenic mixed lymphocyte reaction (MLR) and/or suppressing the cytolytic activity of intestinal intraepithelial lymphocytes (iIELs). The antibody is a human, humanized, xenogeneic, or a chimeric human-murine antibody. The antigen-binding fragment is selected from a single chain Fv fragment, an F(ab') fragment, an F(ab) fragment, and an F(ab')<sub>2</sub> fragment. The ligand-binding molecule is an antibody defined above or its immunoglobulin chain. Preferred Polynucleotide: The polynucleotide of (3) encodes at least a variable region of an immunoglobulin chain of the antibody defined above, where the variable region comprises at least one complementarity determining region (CDR) of the VH and/or VL of the variable region of the antibody. Preparation (claimed): The antibody is prepared by culturing the cell of (5), and isolating the antibody, its functional fragment or immunoglobulin chain(s) from the culture.

Preferred Composition: The composition is a pharmaceutical composition and further comprises a pharmaceutical carrier, and an immunosuppressive agent.

ACTIVITY - Immunomodulator; Immunosuppressive; Cytostatic; Antibacterial; Vulnerary. No biological data given.

MECHANISM OF ACTION - Vaccine; T cell Activator.

USE - The antibody is useful for modulating an immune response, or in the preparation of a pharmaceutical composition for modulating an immune response. The antibody, ligand binding molecule, or its corresponding anti-idiotypic antibody can be used for diagnosing and/or treating a disorder related to the neo-expression or malfunction of BGP(C-CAM1), or for targeting a therapeutic and/or diagnostic agent to a cell which expresses BGP(C-CAM1) or its fragment (all claimed). Specifically, the antibody is useful for treating graft versus host disease, sepsis or tumors; and for improving wound healing.

ADMINISTRATION - The antibody or composition comprising the antibody is administered intravenously, intramuscularly, subcutaneously, intraperitoneally, as an aerosol (claimed), topically or intradermally at a dose of 0.01 microg-10 mg/kg body weight.

EXAMPLE - The 34B1, 26H7 and 5F4 monoclonal antibodies (mAbs) were produced by immunizing a BALB/c mice with the activated human mucosal lymphocyte line 191 E. Three intraperitoneal injections and a final intravenous injection of 5x10<sup>6</sup> lymphocytes were given at 2-week intervals. Three days after intravenous immunization, splenocytes were isolated and fused with NS 1 murine myeloma cells in the presence of PEG. Hybridomas were selected with aminopterin-containing medium, and hybridoma supernatants were screened by indirect immunoperoxidase staining frozen intestinal and tonsillar tissue sections. Positive hybridomas were subcloned twice by limiting dilution, and ascites containing the antibody was produced by intraperitoneal injection of the hybridoma cells into pristane-treated BALB/c mice. Isotypes of 34B1 (IgG1), 26H7 (IgG1), and 5F4 (IgG1) were determined by ELISA using murine isotype-specific mAb. mAbs were purified affinity purification and protein-A sepharose columns by standard methods. (16 pages)

L10 ANSWER 3 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN

Full Text	Citing References
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AN 2002:754149 CAPLUS

DN 137:261887

TI Antibodies and immune complexes for enhancement of the immune response to tumor

IN Schultes, Birgit C.; Nicodemus, Christopher F.

PA Altarex Corp., USA

SO PCT Int. Appl., 111 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2002076384	A2	20021003	WO 2002-US7272	20020308
	WO 2002076384	A3	20030501		
	W:				
	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
	CA 2441393	AA	20021003	CA 2002-2441393	20020308
	GB 2390811	A1	20040121	GB 2003-24503	20020308
	GB 2390811	B2	20060118		
	GB 2413960	A1	20051116	GB 2005-14967	20020308
	US 2005031619	A1	20050210	US 2004-472167	20040402
PRAI	US 2001-277599P	P	20010321		
	GB 2003-24503	A3	20020308		



WO 2002-US7272 W 20020308

AB The authors disclose the use of antibodies and immune complexes to enhance the immunogenicity of the host for tumor antigens.

L10 ANSWER 4 OF 8 Elsevier BIOBASE COPYRIGHT 2006 Elsevier Science

	Full Text	Citing References
	B.V. on STN	
AN	2001059228 ESBIODASE	
TI	Development of a minimally immunogenic variant of humanized anti-carcinoma monoclonal antibody CC49	
AU	Kashmiri S.V.S.; Iwahashi M.; Tamura M.; Padlan E.A.; Milenic D.E.; Schlom J.	
CS	S.V.S. Kashmiri, Laboratory Tumor Immunology/Biology, National Cancer Institute, National Institutes of Health, Bethesda, MD 20892, United States. E-mail: <a href="mailto:sk85f@nih.gov">sk85f@nih.gov</a>	
SO	Critical Reviews in Oncology/Hematology, (2001), 38/1 (3-16), 90 reference(s) CODEN: CCRHEC ISSN: 1040-8428	
PUI	S1040842800001335	
DT	Journal; General Review	
CY	Ireland	
LA	English	
SL	English	
AB	Monoclonal antibody (MAb) CC49 reacts with a pancarcinoma antigen, tumor associated glycoprotein (TAG)-72. To circumvent human anti-murine antibody (HAMA) responses in patients, we earlier developed a humanized CC49 (HuCC49) by grafting the complementarity-determining regions (CDRs) of MAb CC49 onto variable light (VL) and variable heavy (VH) frameworks of the human MABs LEN and 21/28'CL, respectively. With the aim of minimizing its immunogenicity further, we have now generated a variant HuCC49 MAb by grafting the specificity-determining residues (SDRs) of MAb CC49 onto the frameworks of the human MABs. Based on the evaluation of its binding affinity for TAG-72 and its reactivity with anti-idiotypic antibodies present in sera from patients who have been treated with murine CC49, this variant retains its antigen-binding activity and shows minimal reactivity with anti-idiotypic antibodies in patients' sera. Development of this variant, which is a potentially useful clinical reagent for diagnosis and therapy of human carcinomas, demonstrates that for humanization of a <b>xenogeneic antibody</b> grafting of the potential SDRs should be sufficient to retain its antigen-binding properties. Copyright © 2001 Elsevier Science Ireland Ltd.	

L10 ANSWER 5 OF 8 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V. on

	Full Text	Citing References
	STN	
AN	1991:21244954 BIOTECHNO	
TI	Oral immunization with <b>xenogeneic antibodies</b> stimulates the production of systemic and mucosal <b>anti-idiotypic</b> antibodies	
AU	Collins A.M.; Robertson D.M.; Hosking C.S.; Flannery G.R.	
CS	Dept. of Microbiology/Immunol., Univ. of New South Wales, P.O. Box 1, Kensington, NSW 2033, Australia.	
SO	Immunology, (1991), 73/4 (388-393) CODEN: IMMUM ISSN: 0019-2805	
DT	Journal; Article	
CY	United Kingdom	
LA	English	
SL	English	
AB	The humoral and mucosal immune responses to oral immunization with <b>xenogeneic antibodies</b> were studied using an animal model in which female rabbits were fed daily doses of the MOPC-315 murine IgA antibody, and were mated during the course of the feeding programme. Serum and colostrum samples were assayed for the presence of <b>anti-idiotypic</b> antibodies by ELISA assay, before and after depletion of anti-IgA	

antibodies, by affinity chromatography using another murine IgA idiotype. It was shown that all animals responded to exposure to the MOPC-315 idiotype with the production of serum anti-murine immunoglobulin antibodies and that four of six animals produced serum **anti-idiotypic** antibodies. That the immune response included antibodies directed against the antigen-binding site was confirmed by competition ELISA assay. Mucosal IgG and IgA anti-immunoglobulin antibodies were present in milk from all antibody-fed rabbits tested, and IgA **anti-idiotypic** antibodies were detectable in the colostrum of one rabbit. The results provide some support for the hypothesis that human exposure to **xenogeneic antibodies**, most commonly bovine milk immunoglobulins, may provoke the production of **anti-idiotypic** antibodies, and that such exposure may lead to disturbances of immune regulation.

L10 ANSWER 6 OF 8 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V. on

	Full Text	Citing References
	STN	
AN	1988:19044855	BIOTECHNO
TI	Problems and prospects in the use of lymphoma idiotypes as therapeutic targets	
AU	Stevenson G.T.; Glennie M.J.; Hamblin T.J.; Lane A.C.; Stevenson F.K.	
CS	Lymphoma Research Unit, Tenovus Laboratory, General Hospital, Southampton SO9 4XY, United Kingdom.	
SO	International Journal of Cancer, (1988), 42/SUPPL. 3 (9-12) CODEN: IJCNAA ISSN: 0020-7136	
DT	Journal; Article	
CY	United States	
LA	English	
SL	English	
AB	The infusion of <b>anti-idiotypic</b> antibody in patients with lymphoma has proved a relatively innocuous procedure, but in general has yielded only partial, short-lived remissions of disease. A major problem is that <b>xenogeneic antibody</b> is simply not well-fitted to destroying mammalian cells: complement and cellular effectors (K cells and phagocytes) are not efficiently recruited, and the target cells in any case present some excellent defense mechanisms. Antigenic modulation is particularly prominent among these defenses, and we present evidence here for modulation of idiotype being much more efficient in vivo than in vitro. Two broad types of antibody derivative are under development to improve the killing of neoplastic targets. One type relies on recruiting natural effectors, and is exemplified by univalent chimeric antibody. The other relies on delivering an exogenous effector such as a drug or toxin, and is exemplified by bispecific anti-Id/anti-saporin F(ab') <sub>2</sub> antibody. Both types of derivative have been able to suppress animal lymphoma to the extent that tumor escape occurs largely through the emergence of idiotype-negative mutants.	

L10 ANSWER 7 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN

	Full Text	Citing References
AN	1985:594621	CAPLUS
DN	103:194621	
TI	Monoclonal anti-idiotypic antibodies against the murine B cell lymphoma 38C13: characterization and use as probes for the biology of the tumor in vivo and in vitro	
AU	Maloney, David G.; Kaminski, Mark S.; Burowski, Daphna; Haimovich, Joseph; Levy, Ronald	
CS	Med. Sch., Stanford Univ., Stanford, CA, 94305, USA	
SO	Hybridoma (1985), 4(3), 191-209 CODEN: HYBRDY; ISSN: 0272-457X	
DT	Journal	
LA	English	
AB	To establish a murine model for the monoclonal anti-idiotypic (Id) immunotherapy of B cell lymphoma, a panel of rat and murine monoclonal anti-Id antibodies of several different isotypes was generated against the	

surface Ig of the murine B cell tumor 38C13 (38C). **Xenogeneic antibodies** were made from fusions of rat spleen cells immunized with the 38C Id. Syngeneic monoclonal anti-idiotypes were generated from mice immunized with the Id conjugated to keyhole limpet hemocyanin. Small differences were noted in the ability of the antibodies to cross-block one another, but all appeared to be directed against the same or closely spaced idiotopes on the immunoglobulin mol. The antibodies selectively pptd. surface Ig from 38C tumor cells and not from normal mouse spleen cells. They were used to selectively stain 38C tumor cells in cell suspensions for fluorimetric anal. or immunohistochem. staining of tissue sections from mice bearing the tumor. As the malignancy progressed, the no. of tumor cells found in all tissues examd. increased. Thus, the anti-Id antibodies provided a specific probe for tumor cell detection. The antibodies had no detectable effect on cell growth in vitro; however, they did cause the rapid transient loss of the expression of cell surface Ig. This modulation was concn. and time dependent but not 100% complete. Re-expression of the Id occurred by 24 h following removal of the anti-Id antibodies. When these antibodies were used in sensitive radioisotope and enzyme linked immunoassays, the tumor cells were found to secrete small amts. of Id in vitro and in vivo. The level of Id detected in vivo correlated with tumor growth and inversely with survival.

L10 ANSWER 8 OF 8 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V. on

	Full Text	Citing References
	STN	
AN	1983:13060779	BIOTECHNO
TI	Site-directed chemotherapy with a drug bound to <b>anti-idiotypic</b> antibody to a lymphoma cell-surface IgM	
AU	Hurwitz E.; Kashi R.; Burowsky D.; et al.	
CS	Dep. Chem. Immunol., Weizmann Inst. Sci., Rehovot 76100, Israel.	
SO	International Journal of Cancer, (1983), 31/6 (745-748)	
	CODEN: IJCNAW	
DT	Journal; Article	
CY	Switzerland	
LA	English	
AB	<b>Xenogeneic antibodies</b> against the cell-surface IgM of a B-cell lymphoma (38C-13) were coupled through a dextran bridge to the anti-neoplastic drug, daunomycin. The conjugate maintained both its antibody and its drug activity. The effectiveness of the conjugate was tested in vivo in mice challenged with the 38C-13 lymphoma. Drug conjugates of the idiotypic antibodies injected intraperitoneally 2 days after tumor transplantation almost completely inhibited tumor development. The controls, daunomycin-dextran-goat anti-DNP, free daunomycin, or the antibodies alone had no effect or only delayed the tumor development.	

=> s hama and ca 125

L11 38 HAMA AND CA 125

=> s l11 and (idiotype or idiotypic)

L12 9 L11 AND (IDIOTYPE OR IDIOTYPIC)

=> duplicate remove l12

DUPLICATE PREFERENCE IS 'CAPLUS, BIOTECHNO, ESBIODASE'

KEEP DUPLICATES FROM MORE THAN ONE FILE? Y/(N):n

PROCESSING COMPLETED FOR L12

L13 9 DUPLICATE REMOVE L12 (0 DUPLICATES REMOVED)

=> d l13 bib abs 1-9

L13 ANSWER 1 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN

	Full Text	Citing References
AN	1999:811105	CAPLUS

DN 132:49023  
 TI Therapeutic compositions that produce an immune response by altering the antigen  
 IN Madiyalakan, Ragupathy; Schultes, Birgit; Baum, Richard P.; Noujaim, Antoine A.; Leveugle, Beatrice; Kreutz, Fernando T.  
 PA Altarex Corp., Can.  
 SO PCT Int. Appl., 78 pp.  
 CODEN: PIXXD2  
 DT Patent  
 LA English  
 FAN.CNT 9

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	<u>WO 9965517</u>	A2	19991223	<u>WO 1999-IB1114</u>	19990615
	<u>WO 9965517</u>	A3	20000203		
	W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW				
	RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	<u>JP 2001055341</u>	A2	20010227	<u>JP 2000-200702</u>	19960515
	<u>NZ 503032</u>	A	20011130	<u>NZ 1996-503032</u>	19960515
	<u>EP 1297846</u>	A1	20030402	<u>EP 2002-18963</u>	19960515
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, AL				
	<u>PT 910407</u>	T	20030731	<u>PT 1996-913660</u>	19960515
	<u>ES 2193240</u>	T3	20031101	<u>ES 1996-913660</u>	19960515
	<u>ZA 9810275</u>	A	20000612	<u>ZA 1998-10275</u>	19981110
	<u>CA 2333221</u>	AA	19991223	<u>CA 1999-2333221</u>	19990615
	<u>AU 9941593</u>	A1	20000105	<u>AU 1999-41593</u>	19990615
	<u>AU 762699</u>	B2	20030703		
	<u>EP 1085902</u>	A2	20010328	<u>EP 1999-925215</u>	19990615
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
	<u>JP 2002518342</u>	T2	20020625	<u>JP 2000-554395</u>	19990615
	<u>US 2002048586</u>	A1	20020425	<u>US 1999-376604</u>	19990818
	<u>JP 2004002481</u>	A2	20040108	<u>JP 2003-315495</u>	20030908
PRAI	<u>US 1998-94598</u>	A	19980615		
	<u>US 1998-152698</u>	A	19980902		
	<u>EP 1996-913660</u>	A3	19960515		
	<u>JP 1997-540681</u>	A3	19960515		
	<u>JP 2000-200702</u>	A3	19960515		
	<u>NZ 1996-332588</u>	A1	19960515		
	<u>WO 1996-IB461</u>	A2	19960515		
	<u>US 1997-877511</u>	A2	19970617		
	<u>WO 1999-IB1114</u>	W	19990615		

AB The invention concerns methods and compns. for stimulating a host's immune response (cellular and humoral immune response), particularly for the treatment of cancer. The methods and compns. according to the invention use binding agents to generate an immune response to a predetd. sol. antigen, esp. a tumor-assocd. antigen such as CA125, CA19.9 and CA15.3. The binding agent is an antibody, a monoclonal antibody or murine monoclonal antibody that does not induce HAMA (human anti-murine antibodies) in patient or in the host. In accordance with the invention, the binding agent-sol. antigen complex alters the immunogenic condition of the host by generating new immunogens that are recognizable by the immune system. This leads to a humoral and/or cellular immune response.

L13 ANSWER 2 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN

Full Text	Citing References
AN 1998:533699	CAPLUS
DN 129:243768	

TI Interferences with two-site immunoassays by human anti-mouse antibodies formed by patients treated with monoclonal antibodies: comparison of different blocking reagents

AU Reinsberg, Jochen

CS Zentrum fur Frauenheilkunde und Geburtshilfe, Universitat Bonn, Bonn, D-53127, Germany

SO Clinical Chemistry (Washington, D. C.) (1998), 44(8, Pt. 1), 1742-1744  
CODEN: CLCHAU; ISSN: 0009-9147

PB American Association for Clinical Chemistry

DT Journal

LA English

AB The most widely used approach for reducing interferences with two-site immunoassays by human anti-mouse antibodies (**HAMAs**) developed by patients after exposure to murine Ig is to include high amts. of non-specific mouse IgG within the assay buffer. Recently, a polymd. form of murine IgG (MAK33) was reported to be superior to normal mouse IgG for blocking **HAMA** interference. In contrast, the authors obsd. false pos. values for the cancer antigen CA125 because of **HAMA** interference in samples from ovarian cancer patients treated with the anti-CA 125 antibody OC125. This interference could be cor. by preincubation with mouse IgG but not with MAK33. The aim of the present study was to examine whether treatment with other monoclonal antibodies gives rise to addnl. **HAMAs** that are insensitive to MAK33 and to further characterize this specific **HAMA** response.

RE.CNT 16 THERE ARE 16 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 3 OF 9 Elsevier BIOBASE COPYRIGHT 2006 Elsevier Science

Full Text	Citing References
	B.V. on STN
AN 1997202196	ESBIOBASE
TI ACS OV: An alternative to well established tests also measuring CA 125?	ACS OV: Eine Alternative zu etablierten Tests fur die Messung von CA 125?
AU Roth H.-J.; Zahn I.	
CS H.-J. Roth, Laborgruppe Heidelberg, Abt Endokrinol Onkol Radioimmunol, Im Breitspiel 15, D-69126 Heidelberg, Germany. E-mail: <a href="mailto:pablolim@aol.com">pablolim@aol.com</a>	
SO Clinical Laboratory, (1997), 43/6 (515-526), 27 reference(s) CODEN: CLLAFP ISSN: 1433-6510	
DT Journal; Article	
CY Germany, Federal Republic of	
LA German	
SL English; German	
AB The objective of the study was to evaluate the validity of the newly developed test ACS OV on the fully automated chemiluminescence analyser ACS:180 Plus. 246 patient samples with CA 125 values covering the entire measuring range were taken out of the daily laboratory routine and analysed with the ACS OV method and 4 commercially available well established tests also measuring CA 125 in serum and plasma. Samples were stored deep frozen at -20°C until parallel measurement was performed. Additionally, 20 selected patient samples previously measured with Enzymun Test® CA 125 I generation and described as 'falsely elevated with old BMD assay', 6 patient samples with medium and high concentrations of human anti-mouse antibodies ( <b>HAMA</b> ), and 24 samples from apparently healthy laboratory technicians were included. Overall run imprecision of ACS OV with coefficients of variation not exceeding 4.1% was excellent for pool sera and commercially available QC-samples. The good assay precision was also shown by calculation of cumulative histograms for 294 patients samples. For 90% of these the coefficients of variation did not exceed 5% for determination in duplicate. A strong linear correlation was observed between ACS OV and the reference method Centacor CA 125II® with slopes of 0.828 (r = 0.966) and 0.853 (r = 0.897) for the entire measuring range of the reference method and up to 100 U/ml, respectively. Comparability with the competitor tests with coefficients of correlation between r = 0.966 and r = 0.990 was good too.	

ACS OV tended to measure CA 125 values about 15% lower than the reference method across the entire assay range. The overall lower values and an expected cut-off value of about 21 U/ml, based on a small number of healthy control subjects, reveals the need for further investigations to establish decision limits. It should be pointed out that the ACS OV test showed no interference even with very high levels of HAMA and with none of the samples which were 'falsely elevated with old BMD assay'. Due to the selection of B27.1 as capture antibody (OC125 antigenic domain) one can assume as well that circulating anti-idiotypic anti-OC125 antibodies might not interfere in the test. In our estimation the newly developed ACS OV test, with its evaluated test characteristics and its excellent automated technical performance on the fully automated analyser ACS:180 Plus, is a reliable tool for monitoring ovarian cancer patients and provides a good alternative to well established tests also measuring CA 125.

L13 ANSWER 4 OF 9 Elsevier BIOBASE COPYRIGHT 2006 Elsevier Science



B.V. on STN

AN 1995084654 ESBIOBASE  
 TI Determination of the cancer antigen 125 with a second generation assay - Interferences by human anti-OC125 antibodies  
 BESTIMMUNG DES CANCER ANTIGEN 125 MIT DER ZWEITEN TEST-GENERATION - INTERFERENZEN DURCH HUMANE ANTI-OC125-ANTIKORPER  
 AU Reinsberg J.; Richter H.  
 CS Dr. J. Reinsberg, Zentrum Frauenheilkunde Geburtshilfe, Universitat Bonn, Sigmund-Freud-Strasse 25, D-53127 Bonn, Germany.  
 SO Klinisches Labor, (1995), 41/5 (345-350)  
 CODEN: KLLAEA ISSN: 0941-2131  
 DT Journal; Article  
 CY Germany, Federal Republic of  
 LA English  
 SL English; German  
 AB We examined the second generation CA-125 assay Enzymun Test CA-125 II, which involves enzyme-linked OC125 detector antibodies and biotinylated M11 capture antibodies, for interferences due to human anti-OC125 antibodies. Testing serum samples obtained from ovarian cancer patients treated with OC125 fragments, with anti-idiotypic anti-OC125 antibody concentrations ranging from  $4.6 \times 10^3$  to  $2.1 \times 10^6$  kU/l and non-specific human anti-mouse antibody concentrations ranging from 36 to 34830  $\mu\text{g/l}$  we found falsely high CA 125 values: in the presence of anti-mouse antibody concentrations between 180  $\mu\text{g/l}$  and 320  $\mu\text{g/l}$  a falsely high assay response equivalent to a CA-125 concentration of 400 kU/l was measured. After elimination of these interferences by addition of murine IgG, an inhibitory effect of anti-OC125 antibodies became evident: from samples with anti-idiotypic anti-OC125 antibody concentrations exceeding  $4.0 \times 10^4$  kU/l less than 22% of added CA 125 was recovered. The present data suggest that non-specific human anti-mouse antibodies are responsible for falsely high results while the reduction of the recovery rate is due to an inhibition of binding of OC125 detector antibodies to CA-125 by anti-idiotypic anti-OC125 antibodies. With the present test protocol the Enzymun Test CA-125 II should therefore not be used for CA-125 determination in patients treated with OC125 fragments.

L13 ANSWER 5 OF 9 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V. on



STN

AN 1995:25162739 BIOTECHNO  
 TI Antiidiotypic induction therapy: Evidence for the induction of immune response through the idiotypic network in patients with ovarian cancer after administration of anti-CA125 murine monoclonal antibody B43.13  
 AU Madiyalakan R.; Sykes T.R.; Dharampaul S.; Sykes C.J.; Baum R.P.; Hor G.; Noujaim A.A.

CS Biomira Research Inc., 1134 Dentistry Pharmacy Bldg., University of  
Alberta, Edmonton, Alta. T6G 2N8, Canada.  
SO Hybridoma, (1995), 14/2 (199-203)  
CODEN: HYBRDY ISSN: 0272-457X  
DT Journal; Conference Article  
CY United States  
LA English  
SL English  
AB The immune status of ovarian cancer patients receiving anti-CA125 routine  
monoclonal antibody B43.13 was evaluated by measuring antiidiotypic  
antibodies (Ab<sub>2</sub>), antiidiotypic antibodies (Ab<sub>3</sub>),  
antiisotypic human antimouse antibodies (HAMA), interferon-γ, and  
CA125 levels in the serum. A specific assay was developed for the  
determination of Ab<sub>2</sub> antibodies using chimeric MAb B43.13. Of the 50  
patients studied, 26 had elevated levels of Ab<sub>2</sub>. Eleven of these 26  
patients also had high titer of antiidiotypic (Ab<sub>3</sub>) antibodies.  
Eight of the 22 patients analyzed had increased interferon-γ  
levels. A tentative correlation was found between survival of these  
patients' antiidiotypic induction.

L13 ANSWER 6 OF 9 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V. on

Full Text	Citing References
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STN

AN 1994:24046678 BIOTECHNO  
TI Activating anti-**idiotypic** human anti-mouse antibodies for immunotherapy  
of ovarian carcinoma  
AU Baum R.P.; Niesen A.; Hertel A.; Nancy A.; Hess H.; Donnerstag B.; Sykes  
T.R.; Sykes C.J.; Suresh M.R.; Noujaim A.A.; Hor G.  
CS Department of Nuclear Medicine, University Medical Center, Theodor Stern  
Kai 7, 60590 Frankfurt/Main, Germany.  
SO Cancer, (1994), 73/SUPPL. (1121-1125)  
CODEN: CANCAR ISSN: 0008-543X  
DT Journal; Conference Article  
CY United States  
LA English  
SL English  
AB Human anti-mouse antibodies (**HAMA**) are observed frequently after  
immunoscintigraphy with monoclonal antibodies (MoAb) directed against  
**CA- 125**. As the authors have shown previously, **HAMA** can cause  
false-positive **CA- 125** values in routine **CA-125** immunoradiometric  
assay (IRMA) tumor-marker assays (in one case, up to 900 days after  
immunoscintigraphy). In 32 patients, the authors found a **HAMA** frequency  
of 34% (11/32: 3/7 after the first administration, 6/13 after the second,  
and 2/2 after the third). Ten patients developed extremely high  
**CA-125** levels after undergoing the CIS IRMA assay (up to 80,000 U/ml)  
in parallel to a significant **HAMA** increase. The use of different  
assays, or **HAMA** removal before in vitro testing, can solve this  
problem. After a new **CA-125** assay containing antibodies that  
recognize different epitopes on the **CA-125** antigen (Biomira TruQuant  
OV) was applied, only mildly increased assay results or normal levels  
were measured. Most of **HAMA**-positive patients demonstrated a  
predominantly anti-**idiotypic** response, determined with two different  
**HAMA** assays. Seven patients with anti-**idiotypic HAMA** responses  
after OC-125 immunoscintigraphy remained free of tumor or had stable  
disease (2-42 or more months), contrary to their poor prognoses that had  
been made based on the underlying stages of their tumors. All of these  
patients are currently doing well (Karnofsky Index > 70%) and show no  
significant tumor progression. In light of their extremely poor prognoses  
(5- year survival rates of 3-5% in recurrent International Federation of  
Gynecology and Obstetrics III/IV stages), without further chemotherapy,  
these courses are extremely unusual. Preliminary in vitro experiments  
lead to the postulation that anti-**idiotypic HAMA** may trigger an  
antitumor effect either by suppressing the growth of **CA-125**-  
expressing cancer cells directly, or by activating the patient's immune  
response via induction of Ab<sub>3</sub>. Similar results are observed after

immunoscintigraphy with a technetium-99m-labeled anti-CA-125 monoclonal antibody (B43.13), which the authors now also use for immunotherapy of ovarian cancer patients by repeated injections, hoping that induction of anti-idiotypic HAMA will be beneficial for prolonged survival of patients with ovarian carcinoma.

L13 ANSWER 7 OF 9 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V. on

Full Text	Citing References
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STN

AN 1993:24198149 BIOTECHNO  
 TI Clinical course of ovarian cancer patients under repeated stimulation of HAMA using MAb OC125 and B43.13  
 AU Baum R.P.; Noujaim A.A.; Nanci A.; Moebus V.; Hertel A.; Niesen A.; Donnerstag B.; Sykes T.; Boniface G.; Hor G.  
 CS Department of Nuclearmedicine, University of Frankfurt, Theodor Stern Kai 7,W-6000 Frankfurt/Main, Germany.  
 SO Hybridoma, (1993), 12/5 (583-589)  
 CODEN: HYBRDY ISSN: 0272-457X  
 DT Journal; Conference Article  
 CY United States  
 LA English

L13 ANSWER 8 OF 9 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V. on

Full Text	Citing References
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STN

AN 1992:22057371 BIOTECHNO  
 TI Human antibody response to the intravenous and intraperitoneal administration of the F(ab')<sub>2</sub> fragment of the OC125 murine monoclonal antibody  
 AU Maher V.E.; Drukman S.J.; Kinders R.J.; Hunter R.E.; Jennings J.; Brigham C.; Stevens S.; Griffin T.W.  
 CS Division of Oncology/Medicine, University of Massachusetts, Medical Center, 55 Lake Avenue North,Worcester, MA 01655, United States.  
 SO Journal of Immunotherapy, (1992), 11/1 (56-66)  
 CODEN: JOIME7 ISSN: 1053-8550  
 DT Journal; Article  
 CY United States  
 LA English  
 SL English  
 AB We have characterized the human immune response against murine monoclonal antibodies (HAMA) in 18 patients following administration of the F(ab')<sub>2</sub> fragment of the murine monoclonal antibody OC125. OC125 is directed against the CA125 antigen, present on the surface of many human ovarian cancers. An affinity matrix was used to separate serum into immunoglobulin-containing and immunoglobulin-free fractions. HAMA titer was determined on the immunoglobulin fraction with an OC125 sandwich enzyme-linked immunosorbent assay (ELISA). All patients developed an HAMA response, despite the use of F(ab')<sub>2</sub> fragments and small amounts (1-4 mg) of antibody. It may be that the intraperitoneal (i.p.) route provides a more marked HAMA response. Enzyme-linked sandwich immunoassays were also used to determine anti-isotype and anti-idiotypic titers. Anti-isotype titers were analyzed with antigen irrelevant, isotype-matched murine antibodies and OC125-HRPO. Anti-idiotypes titers were assessed in a sandwich assay that utilized F(ab')<sub>2</sub> and F(ab') fragments of OC125. The anti-isotype response tended to be of low titer and short duration, while the anti-idiotypic response was of high titer and remarkably persistent. HAMA interfered in an unpredictable manner with the correct measurement of serum levels of CA125 in an enzyme immunoassay using OC125. Corrected values of CA125 could be obtained by measurement of antigen in the immunoglobulin-free fraction of serum. The response of one patient, who developed a markedly elevated anti-idiotypic titer after serial i.v./i.p. injections, was further characterized and found to contain an antibody consistent with an anti-anti-idiotypic to the CA125 antigen.



L13 ANSWER 9 OF 9 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V. on

Full Text	Citing References
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STN

AN 1990:20251933 BIOTECHNO

TI Interference by human anti-mouse antibodies in CA 125 assay after immunoscintigraphy: Anti-**idiotypic** antibodies not neutralized by mouse IgG but removed by chromatography

AU Turpeinen U.; Lehtovirta P.; Alfthan H.; Stenman U.-H.

CS Helsinki University Central Hospital, Department of Obstetrics and Gynecology, Laboratory, Haartmaninkatu 2, 00290 Helsinki, Finland.

SO Clinical Chemistry, (1990), 36/7 (1333-1338)

CODEN: CLCHAU ISSN: 0009-9147

DT Journal; Article

CY United States

LA English

SL English

AB Falsely increased concentrations of the ovarian carcinoma-associated antigen, CA 125, were measured by a monoclonal antibody (MAV)-based double determinant immunoradiometric assay (IRMA) in patients who developed antibodies to mouse immunoglobulins (IgGs) after receiving injections of the same MAB as is used in the CA 125 IRMA. Addition of undiluted mouse serum or purified mouse IgG to the assay mixture failed to eliminate the falsely increased CA 125 concentrations in most of the samples, owing to the presence of anti-**idiotype** antibody. Because of their anti-**idiotypic** nature, the human anti-mouse antibodies (**HAMAS**) had only little effect on other immunometric assays, and this effect could be completely eliminated by addition of mouse IgG. To eliminate the effect of **HAMA** on the CA 125 assay, we studied the ability of various chromatographic methods to separate the interfering **HAMA** from CA 125. For measuring **HAMA** in serum and chromatographic fractions we developed a time-resolved fluoroimmunoassay. Adequate separation of CA 125 and **HAMA** was achieved by affinity chromatography of patients' sera with solid-phase Protein A, Protein G, cation-exchange chromatography on Mono S, and gel filtration on Superose 6. These results demonstrate that the interference can effectively be removed by rather simple chromatographic procedures.

=&gt; s hama and muc-1

L14 2 HAMA AND MUC-1

=&gt; duplicate remove l14

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L15 2 DUPLICATE REMOVE L14 (0 DUPLICATES REMOVED)

=&gt; s hama and muc-1

L14 2 HAMA AND MUC-1

=&gt; d l15 bib abs 1-2

L15 ANSWER 1 OF 2 BIOTECHDS COPYRIGHT 2006 THE THOMSON CORP. on STN

Full Text
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AN 2005-09748 BIOTECHDS

TI New therapeutic composition comprising a non-radiolabeled binding agent or a binding agent other than HMFG1 that specifically binds to an epitope of soluble and tumor-bound tumor-associated MUC-1, useful in treating tumor in a mammal;

for use in tumor gene therapy

AU MADIYALAKAN R; QI W; SCHULTES B C

PA ALTAREX CORP

PI US 2005048059 3 Mar 2005

AI US 2004-754089 7 Jan 2004

PRAI US 2004-754089 7 Jan 2004; US 1999-149492 18 Aug 1999

DT Patent  
 LA English  
 OS WPI: 2005-181416 [19]  
 AN 2005-09748 BIOTECHDS  
 AB DERWENT ABSTRACT:

NOVELTY - A new therapeutic composition comprises a non-radiolabeled binding agent or a binding agent other than HMFG1 that specifically binds to an epitope of soluble and tumor-bound tumor-associated **MUC-1**.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for: (1) treating a mammal bearing a tumor; (2) a binding agent that binds immunological determinants from amino acid residues of a peptide having the amino acid sequence Asp-Thr-Arg-Pro-Ala-Pro or that binds the same epitope as Alt-1; (3) Alt-1; and (4) treating a mammal bearing a tumor, where the animal has a baseline level of anti-**MUC-1** antibody.

BIOTECHNOLOGY - Preferred Composition: The therapeutic composition comprises a non-radiolabeled binding agent or a binding agent other than HMFG1 that specifically binds to an epitope of soluble and tumor-bound tumor-associated **MUC-1**. The binding agent is not a monoclonal antibody comprising HMPV, VU-3-C6, MF06, VU-11-D1, MF30, BCP8, DF3, BC2, B27.29, VU-3-D1, 7540MR, MF11, Bc4E549, VU-11-E2, M38, E29, GP1.4, 214D4, BC4W154, HMFG-2, C595, Mc5 or A76-A/C7. The binding agent induces an anti-idiotypic response and a cellular immune response in the mammal. The binding agent binds immunological determinants from amino acid residues of a peptide having the amino acid sequence Asp-Thr-Arg-Pro-Ala-Pro or that binds the same epitope as Alt-1. The binding agent is photoactivated. The binding agent is coupled to a photodynamic agent. The photodynamic agents include hypocrellins or hypocrellin derivatives. The epitope comprises an immunological determinant that includes carbohydrate. Preferred Method: Treating a mammal bearing a tumor comprises administering the binding agent or therapeutic composition. The binding agent is administered at a dosage that is the maximum amount of binding agent that does not induce antibody-mediated toxicity or that does not produce ADCC or CDC. The binding agent is administered at a dosage that elicits a HAXA response greater than 200 U/ml or that reduces the level of tumor antigen CA15.3. The HAXA response is a **HAMA** response. The method further comprises irradiating the mammal with a visible light source. The binding agent is administered in the absence or presence of an adjuvant. The binding agent binds both circulating and tumor-bound **MUC-1**. Treating a mammal bearing a tumor, where the animal has a baseline level of anti-**MUC-1** antibody comprises subcutaneously administering the therapeutic composition comprising a binding agent that specifically binds to an epitope of tumor-associated **MUC-1** that causes an increase of at least 3-fold in anti-**MUC-1** antibody compared to the baseline level.

ACTIVITY - Cytostatic. No biological data given.

MECHANISM OF ACTION - Gene therapy.

USE - The therapeutic composition is useful in treating a mammal bearing a tumor (claimed).

ADMINISTRATION - Dosage comprises less than about 8, 3, preferably 0.5-2, 0.5-1.5 or 1 mg/30 kg body weight. The composition is administered via intravenous or subcutaneous route. (All claimed.)

EXAMPLE - No relevant examples given. (20 pages)

L15 ANSWER 2 OF 2 BIOTECHDS COPYRIGHT 2006 THE THOMSON CORP. on STN

Full  
Text

AN 2004-14318 BIOTECHDS  
 TI New binding agent, Alt-1, that binds immunological determinants of **MUC-1**, useful for therapeutically treating a mammal bearing a tumor e.g. breast, colon, esophageal, prostate or pancreatic carcinoma, or multiple myeloma;  
     antibody production via cell culture against **MUC-1** for use in disease therapy  
 AU MADIYALAKAN R  
 PA ALTAREX CORP  
 PI US 6716966 6 Apr 2004

AI US 2000-641833 18 Aug 2000  
 PRAI US 2000-641833 18 Aug 2000; US 1999-149492 18 Aug 1999  
 DT Patent  
 LA English  
 OS WPI: 2004-303095 [28]  
 AN 2004-14318 BIOTECHDS  
 AB DERWENT ABSTRACT:

NOVELTY - A binding agent that binds immunological determinants from amino acid residues of an epitope including carbohydrate and a peptide sequence (I) consisting of amino acid residues 3-8 of an 8-amino acid sequence (SEQ ID NO: 2) peptide, is new.

DETAILED DESCRIPTION - A binding agent that binds immunological determinants from amino acid residues of an epitope including carbohydrate and a peptide sequence (I) consisting of amino acid residues 3-8 of an 8-amino acid sequence (SEQ ID NO: 2) peptide. (I) comprises the amino acid sequence: Asp-Thr-Arg-Pro-Ala-Pro (I) INDEPENDENT CLAIMS are also included for: (1) the antibody Alt-1 produced by the hybridoma having ATCC number PTA-975; and (2) a therapeutic composition comprising a binding agent selected from Alt-1 and the binding agent defined above.

BIOTECHNOLOGY - Preferred Binding Agent: The binding agent is photoactivated. Preferred Method: In treating a mammal bearing a tumor, the binding agent is administered at a dosage that is the maximum amount of binding agent that does not induce antibody-mediated toxicity or that does not produce ADCC or CDC. The binding agent is administered at a dosage that elicits a HAXA response of more than 200 U/ml or more than 2000 U/ml, where the HAXA response is a HAMA response. The binding agent may also be administered at a dosage that reduces the level of tumor antigen CA15.3. The binding agent is administered in the absence or in the presence of an adjuvant. The method further comprises irradiating the mammal with a visible light source. The binding agent binds both circulating and tumor-bound MUC-1.

ACTIVITY - Cytostatic. MT-CB6F1 mouse tumor model administered with MAb-Alt-1 resulted in reduction of tumor burden and greater survival advantage compared with mice treated with PBS. A minimum of 3 intravenous injections prior to inoculation of the tumor cells, and 4 subsequence injections were necessary to demonstrate this effect.

MECHANISM OF ACTION - MUC-1 Inhibitor.

USE - The composition comprising the Alt-1 antibody or the binding agent is useful for therapeutically treating a mammal bearing a tumor (claimed) e.g. breast carcinoma, colon carcinoma, esophageal squamous cell carcinoma, pancreatic carcinoma, prostate carcinoma or multiple myeloma.

ADMINISTRATION - The composition is administered at a dose of less than about 8 mg/30 kg body weight, and the binding agent is administered at a dose of less than 3 mg/30 kg body weight, such as 0.5-2 mg/30 kg body weight, preferably 1 mg/30 kg body weight. The composition or the binding agent is administered intravenously or subcutaneously (claimed).

EXAMPLE - The murine cell or hybridoma that secretes the binding agent was generated by immunizing mice with MUC1, and harvesting and immortalizing the antibody-secreting splenocytes by fusing them with the myeloma cell line SP2/0-Ag14. The desired clone was selected by assaying the secreted antibodies for the ability to bind MUC1, and selected clone was then expanded in a medium. Isotype switching of the clone from an IgM to an IgG was performed, and continued 0.2 microm filtration and dilution using formulation buffer was done. Specific binding of the binding agent to MUC1 expressing breast cancer cells and MUC1 transformed murine cells was demonstrated by FACS, fluorescence microscopy, and other binding assays. The binding agent was then purified from the growth media by standard techniques. (18 pages)

=> s hama and psa or (prostate specific antigen)  
 L16 13610 HAMA AND PSA OR (PROSTATE SPECIFIC ANTIGEN)

=> s hama and psa  
 L17 0 HAMA AND PSA

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=> s hama and (prostate specific antigen)
L18          5 HAMA AND (PROSTATE SPECIFIC ANTIGEN)

=> duplicate remove l18
PROCESSING COMPLETED FOR L18
L19          5 DUPLICATE REMOVE L18 (0 DUPLICATES REMOVED)

=> d l19 bib abd 1-5
'ABD' IS NOT A VALID FORMAT FOR FILE 'CAPLUS'
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The following are valid formats:

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ABS ----- GI and AB
ALL ----- BIB, AB, IND, RE
APPS ----- AI, PRAI
BIB ----- AN, plus Bibliographic Data and PI table (default)
CAN ----- List of CA abstract numbers without answer numbers
CBIB ----- AN, plus Compressed Bibliographic Data
CLASS ----- IPC, NCL, ECLA, FTERM
DALL ----- ALL, delimited (end of each field identified)
DMAX ----- MAX, delimited for post-processing
FAM ----- AN, PI and PRAI in table, plus Patent Family data
FBIB ----- AN, BIB, plus Patent FAM
IND ----- Indexing data
IPC ----- International Patent Classifications
MAX ----- ALL, plus Patent FAM, RE
PATS ----- PI, SO
SAM ----- CC, SX, TI, ST, IT
SCAN ----- CC, SX, TI, ST, IT (random display, no answer numbers;
          SCAN must be entered on the same line as the DISPLAY,
          e.g., D SCAN or DISPLAY SCAN)
STD ----- BIB, CLASS

IABS ----- ABS, indented with text labels
IALL ----- ALL, indented with text labels
IBIB ----- BIB, indented with text labels
IMAX ----- MAX, indented with text labels
ISTD ----- STD, indented with text labels

OBIB ----- AN, plus Bibliographic Data (original)
OIBIB ----- OBIB, indented with text labels

SBIB ----- BIB, no citations
SIBIB ----- IBIB, no citations

HIT ----- Fields containing hit terms
HITIND ----- IC, ICA, ICI, NCL, CC and index field (ST and IT)
          containing hit terms
HITRN ----- HIT RN and its text modification
HITSTR ----- HIT RN, its text modification, its CA index name, and
          its structure diagram
HITSEQ ----- HIT RN, its text modification, its CA index name, its
          structure diagram, plus NTE and SEQ fields
FHITSTR ----- First HIT RN, its text modification, its CA index name, and
          its structure diagram
FHITSEQ ----- First HIT RN, its text modification, its CA index name, its
          structure diagram, plus NTE and SEQ fields
KWIC ----- Hit term plus 20 words on either side
OCC ----- Number of occurrence of hit term and field in which it occurs
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To display a particular field or fields, enter the display field codes. For a list of the display field codes, enter HELP DFIELDS at an arrow prompt (=>). Examples of formats include: TI; TI,AU; BIB,ST; TI,IND; TI,SO. You may specify the format fields in any order and the information will be displayed in the same order as the format specification.

All of the formats (except for SAM, SCAN, HIT, HITIND, HITRN, HITSTR, FHITSTR, HITSEQ, FHITSEQ, KWIC, and OCC) may be used with DISPLAY ACC to view a specified Accession Number.

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ABS ----- GI and AB
ALL ----- BIB, AB, IND, RE
APPS ----- AI, PRAI
BIB ----- AN, plus Bibliographic Data and PI table (default)
CAN ----- List of CA abstract numbers without answer numbers
CBIB ----- AN, plus Compressed Bibliographic Data
CLASS ----- IPC, NCL, ECLA, FTERM
DALL ----- ALL, delimited (end of each field identified)
DMAX ----- MAX, delimited for post-processing
FAM ----- AN, PI and PRAI in table, plus Patent Family data
FBIB ----- AN, BIB, plus Patent FAM
IND ----- Indexing data
IPC ----- International Patent Classifications
MAX ----- ALL, plus Patent FAM, RE
PATS ----- PI, SO
SAM ----- CC, SX, TI, ST, IT
SCAN ----- CC, SX, TI, ST, IT (random display, no answer numbers;
              SCAN must be entered on the same line as the DISPLAY,
              e.g., D SCAN or DISPLAY SCAN)
STD ----- BIB, CLASS

IABS ----- ABS, indented with text labels
IALL ----- ALL, indented with text labels
IBIB ----- BIB, indented with text labels
IMAX ----- MAX, indented with text labels
ISTD ----- STD, indented with text labels

OBIB ----- AN, plus Bibliographic Data (original)
OIBIB ----- OBIB, indented with text labels

SBIB ----- BIB, no citations
SIBIB ----- IBIB, no citations

HIT ----- Fields containing hit terms
HITIND ----- IC, ICA, ICI, NCL, CC and index field (ST and IT)
              containing hit terms
HITRN ----- HIT RN and its text modification
HITSTR ----- HIT RN, its text modification, its CA index name, and
              its structure diagram
HITSEQ ----- HIT RN, its text modification, its CA index name, its
              structure diagram, plus NTE and SEQ fields
FHITSTR ----- First HIT RN, its text modification, its CA index name, and
              its structure diagram
FHITSEQ ----- First HIT RN, its text modification, its CA index name, its
              structure diagram, plus NTE and SEQ fields
KWIC ----- Hit term plus 20 words on either side
OCC ----- Number of occurrence of hit term and field in which it occurs

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To display a particular field or fields, enter the display field codes. For a list of the display field codes, enter HELP DFIELDS at an arrow prompt (=>). Examples of formats include: TI; TI,AU; BIB,ST; TI,IND; TI,SO. You may specify the format fields in any order and the information will be displayed in the same order as the format specification.

All of the formats (except for SAM, SCAN, HIT, HITIND, HITRN, HITSTR, FHITSTR, HITSEQ, FHITSEQ, KWIC, and OCC) may be used with DISPLAY ACC to view a specified Accession Number.

ENTER DISPLAY FORMAT (BIB):bib

L19 ANSWER 1 OF 5 CAPLUS COPYRIGHT 2006 ACS on STN

Full Text	Citing References
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AN 2004:354752 CAPLUS  
 DN 140:355853  
 TI Therapeutic adjuvant activity of xenotypic antibody  
 IN Schultes, Birgit C.; Nicodemus, Christopher F.  
 PA Altarex Corp., Can.; Altarex Medical Corporation  
 SO PCT Int. Appl., 72 pp.  
 CODEN: PIXXD2  
 DT Patent  
 LA English  
 FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004035003	A2	20040429	WO 2003-US33027	20031017
WO 2004035003	A3	20050113		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2504656	AA	20040429	CA 2003-2504656	20031017
AU 2003286463	A1	20040504	AU 2003-286463	20031017
EP 1578445	A2	20050928	EP 2003-777662	20031017
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
US 2005271649	A1	20051208	US 2005-531849	20050715
PRAI US 2002-419332P	P	20021017		
WO 2003-US33027	W	20031017		

L19 ANSWER 2 OF 5 CAPLUS COPYRIGHT 2006 ACS on STN

Full Text	Citing References
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AN 2003:221907 CAPLUS  
 DN 138:234416  
 TI Methods and apparatus for conducting multiple measurements on a sample  
 IN Glezer, Eli N.; Johnson, Kent; Tsionsky, Michael; Kenten, John H.; Debad, Jeff D.; Umek, Robert M.; Eason, Paula Denney; Biebuyck, Hans; Wohlstadter, Jacob N.; Wilbur, James; Sigal, George  
 PA Meso Scale Technologies, LLC, USA  
 SO PCT Int. Appl., 147 pp.  
 CODEN: PIXXD2  
 DT Patent  
 LA English  
 FAN.CNT 3

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003023360	A2	20030320	WO 2002-US28652	20020910
WO 2003023360	A3	20030710		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				

<u>CA 2459893</u>	AA	20030320	<u>CA 2002-2459893</u>	20020910
<u>US 2003113713</u>	A1	20030619	<u>US 2002-238391</u>	20020910
<u>US 2003207290</u>	A1	20031106	<u>US 2002-238960</u>	20020910
<u>US 7063946</u>	B2	20060620		
<u>EP 1436620</u>	A2	20040714	<u>EP 2002-798183</u>	20020910
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK				
<u>JP 2005521032</u>	T2	20050714	<u>JP 2003-527386</u>	20020910
<u>US 2005136048</u>	A1	20050623	<u>US 2005-54672</u>	20050209
<u>US 2005136497</u>	A1	20050623	<u>US 2005-55472</u>	20050209
<u>PRAI US 2001-318284P</u>	P	20010910		
<u>US 2001-318289P</u>	P	20010910		
<u>US 2001-318293P</u>	P	20010910		
<u>US 2002-363498P</u>	P	20020311		
<u>US 2002-409442P</u>	P	20020909		
<u>US 2002-238437</u>	A3	20020910		
<u>WO 2002-US28652</u>	W	20020910		

L19 ANSWER 3 OF 5 CAPLUS COPYRIGHT 2006 ACS on STN

Full Text	Citing References
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AN 2001:833121 CAPLUS  
 DN 135:370635  
 TI Therapeutic application of immune complexes and their presentation by dendritic cells  
 IN Schultes, Birgit; Noujaim, Antoine  
 PA Altarex Corp., Can.  
 SO PCT Int. Appl., 66 pp.  
 CODEN: PIXXD2  
 DT Patent  
 LA English  
 FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
<u>PI</u>	<u>WO 2001085204</u>	A2	20011115	<u>WO 2001-IB1331</u>	20010511
	<u>WO 2001085204</u>	A3	20020822		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	<u>CA 2408549</u>	AA	20011115	<u>CA 2001-2408549</u>	20010511
	<u>US 2002048583</u>	A1	20020425	<u>US 2001-853300</u>	20010511
	<u>US 2002164312</u>	A1	20021107	<u>US 2001-853268</u>	20010511
	<u>US 6689355</u>	B2	20040210		
	<u>EP 1294398</u>	A2	20030326	<u>EP 2001-949830</u>	20010511
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
	<u>JP 2003532687</u>	T2	20031105	<u>JP 2001-581857</u>	20010511
	<u>US 2004191236</u>	A1	20040930	<u>US 2003-683510</u>	20031010
<u>PRAI</u>	<u>US 2000-203635P</u>	P	20000511		
	<u>US 2000-253671P</u>	P	20001128		
	<u>US 2000-253956P</u>	P	20001128		
	<u>US 2001-853268</u>	A1	20010511		
	<u>WO 2001-IB1331</u>	W	20010511		

L19 ANSWER 4 OF 5 CAPLUS COPYRIGHT 2006 ACS on STN

Full Text	Citing References
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AN 2001:833120 CAPLUS  
 DN 135:370634  
 TI Therapeutic application of immune complexes and their presentation by

dendritic cells  
 IN Schultes, Birgit; Noujaim, Antoine; Mann, Dean  
 PA Altarex Corp., Can.  
 SO PCT Int. Appl., 65 pp.  
 CODEN: PIXXD2  
 DT Patent  
 LA English  
 FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	<u>WO 2001085203</u>	A2	20011115	<u>WO 2001-IB1238</u>	20010511
	<u>WO 2001085203</u>	A3	20020822		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	<u>CA 2408547</u>	AA	20011115	<u>CA 2001-2408547</u>	20010511
	<u>US 2002048583</u>	A1	20020425	<u>US 2001-853300</u>	20010511
	<u>US 2002164312</u>	A1	20021107	<u>US 2001-853268</u>	20010511
	<u>US 6689355</u>	B2	20040210		
	<u>EP 1294397</u>	A2	20030326	<u>EP 2001-945568</u>	20010511
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
	<u>JP 2003532686</u>	T2	20031105	<u>JP 2001-581856</u>	20010511
	<u>US 2004191236</u>	A1	20040930	<u>US 2003-683510</u>	20031010
PRAI	<u>US 2000-203635P</u>	P	20000511		
	<u>US 2000-253671P</u>	P	20001128		
	<u>US 2000-253956P</u>	P	20001128		
	<u>US 2001-853268</u>	A1	20010511		
	<u>WO 2001-IB1238</u>	W	20010511		

L19 ANSWER 5 OF 5 CAPLUS COPYRIGHT 2006 ACS on STN

Full Text	Citing References
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AN 1999:811105 CAPLUS  
 DN 132:49023  
 TI Therapeutic compositions that produce an immune response by altering the antigen  
 IN Madiyalakan, Ragupathy; Schultes, Birgit; Baum, Richard P.; Noujaim, Antoine A.; Leveugle, Beatrice; Kreutz, Fernando T.  
 PA Altarex Corp., Can.  
 SO PCT Int. Appl., 78 pp.  
 CODEN: PIXXD2  
 DT Patent  
 LA English  
 FAN.CNT 9

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	<u>WO 9965517</u>	A2	19991223	<u>WO 1999-IB1114</u>	19990615
	<u>WO 9965517</u>	A3	20000203		
	W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW				
	RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	<u>JP 2001055341</u>	A2	20010227	<u>JP 2000-200702</u>	19960515
	<u>NZ 503032</u>	A	20011130	<u>NZ 1996-503032</u>	19960515
	<u>EP 1297846</u>	A1	20030402	<u>EP 2002-18963</u>	19960515



R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,  
IE, SI, LT, LV, FI, RO, MK, AL

<u>PT 910407</u>	T	20030731	<u>PT 1996-913660</u>	19960515
<u>ES 2193240</u>	T3	20031101	<u>ES 1996-913660</u>	19960515
<u>ZA 9810275</u>	A	20000612	<u>ZA 1998-10275</u>	19981110
<u>CA 2333221</u>	AA	19991223	<u>CA 1999-2333221</u>	19990615
<u>AU 9941593</u>	A1	20000105	<u>AU 1999-41593</u>	19990615
<u>AU 762699</u>	B2	20030703		
<u>EP 1085902</u>	A2	20010328	<u>EP 1999-925215</u>	19990615

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,  
IE, FI

<u>JP 2002518342</u>	T2	20020625	<u>JP 2000-554395</u>	19990615
<u>US 2002048586</u>	A1	20020425	<u>US 1999-376604</u>	19990818
<u>JP 2004002481</u>	A2	20040108	<u>JP 2003-315495</u>	20030908
<u>PRAI US 1998-94598</u>	A	19980615		
<u>US 1998-152698</u>	A	19980902		
<u>EP 1996-913660</u>	A3	19960515		
<u>JP 1997-540681</u>	A3	19960515		
<u>JP 2000-200702</u>	A3	19960515		
<u>NZ 1996-332588</u>	A1	19960515		
<u>WO 1996-IB461</u>	A2	19960515		
<u>US 1997-877511</u>	A2	19970617		
<u>WO 1999-IB1114</u>	W	19990615		

=> s alterex  
L20 0 ALTEREX

=> s altarex  
L21 15 ALTAREX

=> s l21 and antibody  
L22 13 L21 AND ANTIBODY

=> duplicate remove l22  
DUPLICATE PREFERENCE IS 'CAPLUS, BIOTECHNO, ESBIODBASE'  
KEEP DUPLICATES FROM MORE THAN ONE FILE? Y/(N):n  
PROCESSING COMPLETED FOR L22  
L23 10 DUPLICATE REMOVE L22 (3 DUPLICATES REMOVED)

=> duplicate remove l21  
DUPLICATE PREFERENCE IS 'CAPLUS, BIOTECHNO, ESBIODBASE'  
KEEP DUPLICATES FROM MORE THAN ONE FILE? Y/(N):n  
PROCESSING COMPLETED FOR L21  
L24 12 DUPLICATE REMOVE L21 (3 DUPLICATES REMOVED)

=> d l24 bib abs 1-12

L24 ANSWER 1 OF 12 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 1

	Full Text	Citing References
AN	2004:520066	CAPLUS
DN	141:312437	
TI	Immunotherapy of ovarian cancer with antibodies: a focus on oregovomab	
AU	Berek, Jonathan S.	
CS	24-137 UCLA Center for the Health Sciences, David Geffen School of Medicine at UCLA, Los Angeles, CA, 90095-1740, USA	
SO	Expert Opinion on Biological Therapy (2004), 4(7), 1159-1165 CODEN: EOBTA2; ISSN: 1471-2598	
PB	Ashley Publications Ltd.	
DT	Journal; General Review	
LA	English	
AB	A review. Recent advances in the mol. and cellular biol. of malignancy and tumor immunol. have stimulated significant progress in the application of immunotherapies as adjuvant treatments in cancer. Oregovomab (OvaRex, AltaRex) is a murine monoclonal antibody with high affinity to	

the ovarian cancer assocd. antigen CA125. Infusion of low-dose antibody results in formation of circulating immune complexes which can trigger a cellular immune response targeting CA125 and the ovarian cancer. Oregovomab has activity following initial chemotherapy and in recurrent disease settings and is in Phase III trials to establish its efficacy to prolong time to relapse in patients with advanced ovarian cancer and favorable outcomes to their front-line treatment. Addnl. studies of antigen processing and combination chemo-immunotherapy are ongoing. The treatment shows promise as a potential new addn. to the std. care of ovarian cancer.

RE.CNT 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 2 OF 12 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V.



on STN

AN 2003:36553208 BIOTECHNO  
TI Cancer Drug Development: New Directions and Challenges - SMi Conference:  
10-11 March 2003, London, UK  
AU Erlich R.  
CS R. Erlich, Thomson Current Drugs, Middlesex House, 34-42 Cleveland  
Street, London W1T 4LB, United Kingdom.  
E-mail: [rebecca.erlich@current-drugs.com](mailto:rebecca.erlich@current-drugs.com)  
SO IDrugs, (01 APR 2003), 6/4 (331-333)  
CODEN: IDRUFN ISSN: 1369-7056  
DT Journal; Conference Article  
CY United Kingdom  
LA English  
SL English  
AB Over the last five years, the explosion in knowledge at the molecular level in areas such as signal transduction and the cell cycle has revealed a plethora of molecular targets potentially involved in the pathogenesis of cancer. Cancer drug development is now reflecting the emerging molecular archaeology of the disease and lessons are already being learned from targeted therapies, such as gefitinib (Iressa; AstraZeneca plc), that have not achieved the expected benefits when used in combination with conventional chemotherapy in late-stage clinical trials. The disappointment with Iressa, a therapy that inhibits EGFR, has highlighted the need to rethink clinical trial designs and target populations that will benefit from such treatments. Clinical success with Celgene Corp's thalidomide (Thalomid) has demonstrated that lack of response in animals cannot reliably be extrapolated to predict the efficacy of a drug in patients. However, in the current arena of cancer therapy, it is highly unusual for a drug to reach the market where clinical responses have not been observed in phase I trials, even though this endpoint is not strictly a defining characteristic of the classic phase I trial. To achieve the full benefit from clinical trials with targeted therapies, it will be necessary to demonstrate the activity of the drug through the definition of biomarkers that are present in blood or urine and are therefore easily accessible for assay. A theme echoed throughout the 2 days of the meeting was the necessity to validate biomarkers at the preclinical stage, followed by the need to develop assays that work in the patient. Karol Sikora (AstraZeneca plc, UK), who chaired day one of the meeting proceedings, pointed out that new style phase I trials must find the dose of a drug that inhibits its target, and that the development of the technology to do so will require closer collaborations between academia, biotechnology and 'big pharma'.

L24 ANSWER 3 OF 12 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V.



on STN

AN 2003:36886520 BIOTECHNO  
TI Immune responses to murine monoclonal antibody-B43.13 correlate with prolonged survival of women with recurrent ovarian cancer

AU Mobus V.J.; Baum R.P.; Bolle M.; Kreienberg R.; Noujaim A.A.; Schultes B.C.; Nicodemus C.F.  
 CS Dr. V.J. Mobus, Dept. of Obstetrics and Gynecology, Stadtische Kliniken, Gotenstrasse 6-8, 65929 Frankfurt, Germany.  
 E-mail: [MoebusVolk@aol.com](mailto:MoebusVolk@aol.com)  
 SO American Journal of Obstetrics and Gynecology, (01 JUL 2003), 189/1 (28-36), 23 reference(s)  
 CODEN: AJOGAH ISSN: 0002-9378  
 DT Journal; Article  
 CY United States  
 LA English  
 SL English  
 AB OBJECTIVE: We evaluated the therapeutic efficiency of the murine monoclonal antibody-B43.13 in the treatment of patients with recurrent ovarian cancer. STUDY DESIGN: This was a retrospective study of immune responses and survival in 44 patients who were treated with technetium 99m-labeled monoclonal antibody-B43.13, a murine monoclonal antibody that is directed against the tumor-associated antigen CA125. Most patients were pretreated heavily. Biologic activity was quantified by the assay of immune responses to the human anti-murine antibodies against the monoclonal antibody-B43.13 variable region (Ab<sub>2</sub>) and antibodies that target the CA 125 antigen itself (anti-CA 125 antibody). RESULTS: More than one half of patients (56.8%) survived for >12 months after the first dose of monoclonal antibody B43.13; 34.1% of the patients survived >24 months. To date, 6 of the 44 patients are alive, with survival times of 4 to 7.5 years after the start of the antibody treatment. More than 60% of the evaluable patients met predefined criteria for robust, treatment-emergent human anti-murine antibodies and Ab<sub>2</sub> responses, and these responses were associated with improved survival rates. Median survival time increased approximately 3-fold for human anti-murine antibody responders (22.6 months) versus nonresponders (7.2 months; P < .0016, log-rank test) and 2-fold for Ab<sub>2</sub> responders (18.3 months) versus nonresponders (9.3 months). No serious drug-associated adverse events were reported. CONCLUSION: The associations between multiple types of immune response and improved clinical outcomes suggest that monoclonal antibody-B43.13 should be further evaluated for potential use as an immunotherapy for CA125-expressing malignancies.

L24 ANSWER 4 OF 12 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V.

	Full Text	Citing References
	on STN	
AN	2002:35448064	BIOTECHNO
TI	Monoclonal antibodies as therapeutics in oncology	
AU	Trikha M.; Yan L.; Nakada M.T.	
CS	M. Trikha, Centocor Oncology Research, 200 Great Valley Parkway, Malvern, PA 19355, United States. E-mail: <a href="mailto:mnakada@cntus.jnj.com">mnakada@cntus.jnj.com</a>	
SO	Current Opinion in Biotechnology, (01 DEC 2002), 13/6 (609-614), 29 reference(s) CODEN: CUOBE3 ISSN: 0958-1669	
DT	Journal; General Review	
CY	United Kingdom	
LA	English	
SL	English	
AB	The specificity of antibodies has been harnessed to target cancer cells and the first therapeutic antibodies for use in oncology are now finding application in the clinic. Studies are currently under way to develop new and improved antibodies. Recent developments have been made in the identification of novel targets, including the use of genomic and proteomic technologies. Several methods are also being developed to enhance antibody efficacy.	

L24 ANSWER 5 OF 12 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V.

	Full Text	Citing References
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on STN  
 AN 2002:36470165 BIOTECHNO  
 TI Nature's best weapons to fight cancer. Revival of human monoclonal IgM antibodies  
 AU Vollmers H.P.; Brandlein S.  
 CS Prof. H.P. Vollmers, Institut fur Pathologie, Universitat Wurzburg, Josef-Schneider-Str. 2, D-97080 Wurzburg, Germany.  
 E-mail: [path027@mail.uni-wuerzburg.de](mailto:path027@mail.uni-wuerzburg.de)  
 SO Human Antibodies, (2002), 11/4 (131-142), 123 reference(s)  
 CODEN: HUANFP ISSN: 1093-2607  
 DT Journal; Article  
 CY Netherlands  
 LA English  
 SL English  
 AB The unique features of monoclonal antibodies (specificity, effectiveness, purity and unlimited reproducibility) make them ideal tools for the specific treatment of all kind of diseases. The third generation of monoclonal antibodies for the treatment of human diseases will be, after murine and "humanised" murine immunoglobulins, fully human antibodies. The best source of human monoclonal antibodies are the antibody pools of cancer patients themselves with the best technique for generating them being conventional human hybridoma technology. This technique, will generate human monoclonal antibodies which will not only define important new targets on cancerous tissue, but will also provide the necessary therapeutic human antibodies in the fight against cancer.

L24 ANSWER 6 OF 12 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V.

	Full Text	Citing References
	on STN	
AN	2002:36686878	BIOTECHNO
TI	Immunomodulation with antibodies: Clinical application in ovarian cancer and other malignancies	
AU	Nicodemus C.F.; Schultes B.C.; Hamilton B.L.	
CS	Dr. C.F. Nicodemus, AltaRex Corp., 610 Lincoln Street, Waltham, MA 02451, United States. E-mail: <a href="mailto:cnicodemus@altarex.com">cnicodemus@altarex.com</a>	
SO	Expert Review of Vaccines, (2002), 1/1 (35-48), 95 reference(s) CODEN: ERVXAX ISSN: 1476-0584	
DT	Journal; General Review	
CY	United Kingdom	
LA	English	
SL	English	
AB	This review identifies the role of antibodies in the field of therapeutic cancer vaccines. Ovarian cancer is used as a model to review advances in therapeutic vaccine development with a focus on antibodies as immunomodulators and antigen mimetics, highlighting research on B43.13 and ACA 125. The interaction of biological immunomodulation and chemotherapy is discussed. Requirements of antigen processing and recent advances in the field of dendritic cell biology are critical to current understanding of potent immune response induction. Future directions including use of growth factors, adjuvants and cellular therapies to enhance effects may potentiate observed effects and provide clues for application in many cancers and beyond oncology.	

L24 ANSWER 7 OF 12 CAPLUS COPYRIGHT 2006 ACS on STN

	Full Text	Citing References
AN	2001:370629	CAPLUS
DN	136:68257	
TI	OvaRex ( <a href="http://altarex.com">altarex</a> )	
AU	Brukner, Ivan	
CS	SignalGene Inc, Montreal, QC, H2M 2N9, Can.	
SO	IDrugs (2001), 4(4), 457-462 CODEN: IDRUFN; ISSN: 1369-7056	
PB	Current Drugs Ltd.	

DT Journal; General Review

LA English

AB A review. **AltaRex** is developing OvaRex (B43.13), a monoclonal antibody vaccine, for the potential treatment of ovarian cancer. Addnl., immunotherapy may be possible in other cancers expressing the CA125 antigen, such as breast and lung cancers. **AltaRex** plans to file a BLA for OvaRex with the FDA in late 2001 and for Canadian and European regulatory submissions thereafter, with possible commercialization in 2002 [365081,377828,398528]. In Dec. 2000, **AltaRex** engaged US Oncol. to participate in the company's phase II trial for the "watchful waiting" stage of ovarian cancer. The US Oncol. relationship will bring over 20 satellite sites to the OvaRex study, in addn. to 13 sites that had already begun enrolling patients [394604,384676,365081]. In 1997, **AltaRex** commenced a multicenter, placebo-controlled, double-blind, randomized phase IIb trial in the US for advanced ovarian cancer [230067,331744,344248]. In the following year, **AltaRex** submitted the IND to the FDA for this trial [303667]. Complete anal. of the data is expected by the first half of 2001 [291312,310071,344248]. If an NDA is filed before the end of 2001, approval by mid-2002 is possible [344248]. OvaRex has been awarded Fast Track designation for advanced ovarian cancer [310071] and Orphan Drug status in the US [230067].

RE.CNT 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 8 OF 12 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 2

Full Text	Citing References
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AN 1999:222222 CAPLUS

DN 131:72456

TI Immunotherapy of human ovarian carcinoma with OvaRex MAb-B43.13 in a human-PBL-SCID/BG mouse model

AU Schultes, Birgit C.; Zhang, Chengsheng; Xue, Lanny Y.; Noujaim, Antoine A.; Madiyalakan, Ragupathi

CS AltaRex Corp., Edmonton, AB, T6G 2N8, Can.

SO Hybridoma (1999), 18(1), 47-55

CODEN: HYBRDY; ISSN: 0272-457X

PB Mary Ann Liebert, Inc.

DT Journal

LA English

AB The monoclonal antibody (MAb) B43.13, binding to the ovarian cancer-assocd. antigen CA125, has been injected into >200 patients with ovarian cancer to detect recurrence of the disease. The follow-up of the patients revealed surprisingly long survival spans for several patients despite high CA125 levels. To investigate the therapeutic effectiveness of OvaRex MAb-B43.13 (**AltaRex**, Edmonton, Canada) under well-controlled conditions, the antibody was tested in a human-PBL-SCID/BG mouse model with CA125 pos. human ovarian cancer cells. Mice were reconstituted with human peripheral blood lymphocytes (PBL, normal donors) by i.p. (IP) injection of 2-3x10<sup>7</sup> PBL/mouse. OvaRex MAb-B43.13 was administered at 100 µg/mouse in phosphate buffered saline (PBS), in 3 different exptl. set-ups. An isotype-matched control antibody (MOPC21 or MAb-170) and PBS injection served as controls. The ovarian cancer cell line NIH:OVCAR-NU-3 was injected IP at 1x10<sup>6</sup> cells/mouse or s.c. (SC) at 4x10<sup>6</sup> cells/mouse. Human-PBL-SCID/BG mice were either immunized before injection of tumor cells, along with tumor cells or after small tumors were established (2 wk after transplantation). Antibody injections were repeated twice in 2-wk intervals. Functional and cellular characterization of serum and PBL from these mice demonstrated the successful engraftment of a human immune system in those mice. All 3 expts. showed that OvaRex MAb-B43.13 treatment could (1) delay or prevent development of tumors; (2) reduce the size of small established tumors (SC tumor injection) or suppress ascites formation; (3) delay tumor growth when injected prior to tumor implantation; and (4) prolong the survival of the mice (IP tumor injection).

RE.CNT 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 9 OF 12 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V.

	Full Text	Citing References
	on STN	
AN	1998:28384465	BIOTECHNO
TI	Summary report on the ISOBM TD-6 workshop: Analysis of 20 monoclonal antibodies against Sialyl Lewis(a) and related antigens. Montreux, Switzerland, September 19-24, 1997	
AU	Rye P.D.; Bovin N.V.; Vlasova E.V.; Molodyk A.A.; Baryshnikov A.; Kreutz F.T.; Garinther W.I.; Schultes B.C.; Noujaim A.A.; Madiyalakan R.; Magnani J.; Nilsson O.; Nilsson K.; Nustad K.; Norum L.; Bells H.; Caoh Y.; Suresh M.R.; Very D.L.; Freeman J.V.; Yeung K.K.; Hilgers J.	
CS	Dr. N.V. Bovin, Shemyakin Inst. Bioorganic Chemistry, Russian Academy of Sciences, Ul. Miklukho-Maklaya, 16/10, 117871 GSP-7 Moscow V437, Russian Federation.	
SO	Tumor Biology, (1998), 19/5 (390-420), 13 reference(s) CODEN: TUMBEA ISSN: 1010-4283	
DT	Journal; Article	
CY	Switzerland	
LA	English	
SL	English	
AB	The ISOBM TD-6 Workshop is the first international workshop on monoclonal antibodies against the Sialyl Lewis(a) (SLe(a)) antigen. Eight research groups participated in a blind study to characterize the epitope binding, relative affinity and performance in immunoradiometric assays, of a panel of 20 monoclonal antibodies. The antibodies were tested against a diverse panel of neoglycoconjugates, purified antigens and human serum pools from gastrointestinal malignancies. Epitope specificities were determined for the majority of antibodies in the panel. Cross-reactivity with related saccharide structures was noted in several antibodies. Overall, the results of the TD-6 Workshop show further development of SLe(a) immunoassays may yield yet more specific assays for the detection and management of gastrointestinal and other malignancies.	

L24 ANSWER 10 OF 12 CAPLUS COPYRIGHT 2006 ACS on STN

	Full Text	Citing References
AN	1999:506028	CAPLUS
DN	132:90130	
TI	The effects of circulating antigen on the pharmacokinetics and radioimmunoscintigraphic properties of 99mTc labelled monoclonal antibodies in cancer patients	
AU	McQuarrie, S. A.; Riauka, T.; Baum, R. P.; Sykes, T. R.; Noujaim, A. A.; Boniface, G.; MacLean, G. D.; McEwan, A. J. B.	
CS	Fac. Pharmacy and Pharmaceutical Sci., Univ. Alberta, Edmonton, Can.	
SO	Journal of Pharmacy & Pharmaceutical Sciences [Electronic Publication] (1998), 1(3), 115-125 CODEN: JPPSFY; ISSN: 1482-1826 URL: <a href="http://www.ualberta.ca/~csps/JPPS1(3)/S.McQuarrie/Mabs-McQuarrie.pdf">http://www.ualberta.ca/~csps/JPPS1(3)/S.McQuarrie/Mabs-McQuarrie.pdf</a>	
PB	Canadian Society for Pharmaceutical Sciences	
DT	Journal; (online computer file)	
LA	English	
AB	This article reports the pharmacokinetics, radiation dosimetry and radioimmunoscintigraphy (RIS) of two 99mTc-labeled monoclonal antibodies (Mab) used to detect cancer. The effects of circulating antigen in female cancer patients are explored and their effects on the ability of these MABs to effectively perform as RIS agents noted. To illustrate the effects of circulating antigen, data using Mab B43.13 (OVAREX, AltaRex Corp., Waltham, MA, USA) from a Pilot study in ovarian cancer patients are presented. The results from a Phase II study of Mab 170H.82 (Tru-Scint AD, BIOMIRA INC., Edmonton, Alberta, Canada) in patients with primary and locally recurrent breast cancer were used to portray the biodistribution patterns when no circulating antigen is present. Data from planar gamma camera images were obtained for both groups and used for pharmacokinetic and radiation dosimetry analyses. A pharmacokinetic anal. indicated a	

shorter residence time and higher clearance of  $^{99m}\text{Tc}$ -MAB-B43.13 that was ascribed in part to the circulating CA 125 antigen in this group of ovarian cancer patients. These clearance patterns resulted in acceptable, though higher radiation doses to the spleen and urinary bladder wall for these patients when compared to the MAB-170H.82 group. Both MABs were found to produce acceptable radioimmunoscintigraphic images.

RE.CNT 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 11 OF 12 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V.

Full Text	Citing References
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on STN

AN 1997:27360022 BIOTECHNO

TI Radiolabeling of monoclonal antibody B43.13 with Rhenium-188 for immunoradiotherapy

AU Sykes T.R.; Somayaji V.V.; Bier S.; Woo T.K.; Kwok C.S.; Snieckus V.; Noujaim A.A.

CS T.R. Sykes, Pharmacy/Pharmaceutical Sci. Faculty, University of Alberta, Edmonton, Alta., Canada.

SO Applied Radiation and Isotopes, (1997), 48/7 (899-906), 35 reference(s)  
CODEN: ARISEF ISSN: 0969-8043

PUI S0969804397000250

DT Journal; Article

CY United Kingdom

LA English

SL English

AB In this study we report a novel method for direct radiolabeling of monoclonal antibody B43.13 (MAB-B43.13) with  $^{188}\text{Re}$  and have evaluated the product's radiochemical of biochemical, immunochemical and selected biological properties.  $^{188}\text{Re}$  - MAB - B43.13 was readily prepared by the addition of generator produced perrhenate to a preformulated antibody vial after an optima amount of supplemental stannous ion, in the form of stannous tartrate, was added. The final radiolabeled, product retained its biochemical purity (as determined by size-exclusion HPLC and R/NR-SDS-PAGE), its immunoreactivity (as determined by immunoassay) and presented with a typical stability (in the presence of serum and cysteine) and biodistribution (in tumored mice) profile. The evaluation of the product for immunoradiotherapy of ovarian cancer in a clinical setting requires further studies.

L24 ANSWER 12 OF 12 CAPLUS COPYRIGHT 2006 ACS on STN

Full Text	Citing References
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AN 1997:177231 CAPLUS

DN 126:213678

TI Porous exchanger and water vapor pump: The ALTAREX boiler

AU Guillet, R.; Brunel, G.; Grehier, A.; Viltard, J. C.

CS Gaz de France, Fr.

SO Proceedings of the International Gas Research Conference (1996), Volume Date 1995, (Vol. 2), 2783-2792  
CODEN: PGRCDV; ISSN: 0736-5721

PB Government Institutes

DT Journal

LA English

AB A porous membrane exchanger in the ALTAREX boiler constitutes a new technol. approach to water vapor pumps. This mass and heat exchanger uses the combustion air as a secondary heat sink for recycling the residual latent and sensible energy which is usually lost up the chimney in combustion-type heaters. This means that the ALTAREX boiler can recover all of the higher calorific value of natural gas. In addn. to an increased energy efficiency, the boiler offers the advantages of combustion with high water-vapor enriched air, which generates very little NOx formation. This energy efficiency and low pollution make the ALTAREX boiler and other spin-off technol. systems (i.e., dryers, direct contact heaters, gas turbines, etc.) world-wide leaders in heat energy

prodn. from fuel combustion, esp. natural gas.

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=> s Noujaim, Antoine
L25      0 NOUJAIM, ANTOINE

=> s baum, richard
L26      0 BAUM, RICHARD

=> s (antoine noujaim)
L27      0 (ANTOINE NOUJAIM)

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